CLINICAL CASE

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Ruptured giant *hyperreactio luteinalis* as a life-threatening complication of gestational trophoblastic disease

Hyperreactio Luteinalis gigante rota como complicación potencialmente mortal de la enfermedad trofoblástica gestacional

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ABSTRACT
Hyperreactio luteinalis (HL) is a benign ovarian condition characterized by the presence of multiple thecalutein cysts secondary to elevated levels of human chorionic gonadotropin (hCG); it is a rare complication of the post-molar period. We present the case of a 33-year-old female patient who, four weeks after evacuation of a hydatidiform mole, was admitted with acute abdominal pain and severe hypovolemic shock. An emergency exploratory laparotomy was performed, revealing massive hemoperitoneum (3,000 mL) and rupture of bilateral thecalutein cysts measuring 25 cm. Histopathology confirmed ruptured hydatidiform mole and ruled out active trophoblastic neoplasia. This case demonstrates that the risk of rupture persists after evacuation, requiring continuous monitoring and radical surgery in the event of hemodynamic collapse.
Keywords. Ovarian cysts, Hydatidiform mole, Gestational trophoblastic disease, Hypovolemic shock, Acute abdomen.

RESUMEN
La *hyperreactio luteinalis* (HL) es una condición ovárica benigna caracterizada por la presencia de múltiples quistes tecaluteínicos secundarios a niveles elevados de gonadotropina coriónica humana (hCG), siendo una complicación infrecuente del periodo post-molar. Se presenta el caso de una paciente de 33 años que, cuatro semanas después de la evacuación de una mola hidatiforme, ingresó con abdomen agudo y choque hipovolémico severo, por lo cual se realizó una laparotomía exploratoria de emergencia que reveló un hemoperitoneo masivo de 3000 mL y ruptura de quistes tecaluteínicos bilaterales de 25 cm. La histopatología confirmó HL rota y descartó neoplasia trofoblástica activa. El caso demuestra que el riesgo de ruptura persiste post-evacuación, requiriendo vigilancia continua y cirugía radical ante colapso hemodinámico.
Palabras Clave. Quistes ováricos, Mola hidatiforme, Enfermedad trofoblástica gestacional, Choque hipovolémico, Abdomen agudo.

Introduction

Hyperreactio luteinalis (HL), characterized by the presence of thecaluteinal cysts, is a benign, functional ovarian condition.

These masses arise from hyperstimulation of the inner theca cells, triggered by abnormally elevated serum levels of human chorionic gonadotropin (hCG) or by increased ovarian sensitivity to prolonged hormonal exposure[1]. Clinically, HL manifests as massive ovarian enlargement, typically bilateral, with multiple multilocular cysts filled with clear or serosanguineous fluid[2,3]. Although HL can occur in uncomplicated pregnancies, it is frequently associated with hyperhormonal states such as gestational trophoblastic disease (GTD)[4], multiple pregnancies[5], hyperthyroidism[6], or assisted reproductive technology procedures[7].

GTD encompasses a spectrum of disorders resulting from abnormal trophoblastic proliferation. Its most common form, hydatidiform mole, has an incidence of approximately 1 in every 1,000 to 1,200

pregnancies[8]; it is estimated that about 20% of patients diagnosed with hydatidiform mole develop thealutein cysts due to massive hCG production[4,8,9]. Although hydatidiform mole is usually asymptomatic and is detected incidentally by obstetric ultrasound, large cysts can cause severe abdominal pain, distension, dyspnea, or maternal virilization[2,3,10]. When cysts reach large sizes (more than 15 cm), the risk of acute complications such as torsion, spontaneous rupture, and massive hemoperitoneum increases significantly[2,9]. Transvaginal ultrasound is the first-line diagnostic tool, revealing a characteristic anechoic multilocular "spoke-wheel" pattern; similarly, computed tomography or magnetic resonance imaging are essential for ruling out malignant neoplasms[1,9].

Numerous publications highlight the severity of these complications, as well as the heterogeneity of their presentations; reports such as that by Kurakula et al. describe the rupture of thecalutein cysts as early as 10 weeks' gestation, requiring immediate surgical intervention[11]. Likewise, cases such as that described by Rathod et al. have been reported, highlighting the occurrence of acute abdomen due to cystic rupture specifically in the post-evacuation period following the removal of a hydatidiform mole, demonstrating that the risk of complications persists despite initial treatment of GTD[4]. The management of hydatidiform mole is primarily conservative and relies on the spontaneous resolution of the cysts following the removal of the hCG stimulus (for example, after evacuation of the mole or delivery)[8,10]; however, surgical intervention becomes imperative in the face of acute complications, such as cystic rupture or hypovolemic shock, to ensure hemodynamic stability[1,12]. This report describes an unusual presentation of HL with potentially fatal complications following a molar pregnancy.

Case presentation

A 33-year-old female patient (G2P1) with no significant medical history. During a routine obstetric ultrasound performed at 8 weeks of amenorrhea, findings consistent with a hydatidiform mole were identified (Figure 1); she had serum levels of the beta fraction of human chorionic gonadotropin (β-hCG) of 207,150 IU/L. Management consisted of uterine evacuation via vacuum aspiration, after which she was discharged with strict instructions for weekly β-hCG monitoring and contraception using a progestin implant.

The patient did not comply with the follow-up protocol and returned to the emergency department four weeks after the evacuation, reporting severe abdominopelvic pain and dyspnea. In addition, it was noted that she had begun using combined oral contraceptives without a prescription. Her vital signs indicated severe hypovolemic shock: blood pressure of 78/46 mmHg (mean arterial pressure of 57 mmHg), heart rate of 132 bpm, respiratory rate of 32 bpm, temperature of 35.2 °C, and prolonged capillary refill time (> 3 seconds).

Laboratory results at admission confirmed severe anemia (Hb 6.8 g/dL) and profound metabolic acidosis with respiratory compensation (pH 7.21, HCO_3 10 mmol/L, base excess BE -12 mmol/L), along with an elevated lactate level (3 mmol/L). Serum β-hCG levels remained at 24,100 IU/L (Table 1). A contrast-enhanced abdominal-pelvic CT scan identified bilateral adnexal masses consistent with giant thecalutein cysts, each approximately 25 cm in size, characterized by multiple thin-walled loculi and associated with massive hemoperitoneum (Figure 2).

Due to hemodynamic instability and acute hemorrhagic abdomen, an emergency exploratory laparotomy was performed. Intraoperative findings included 3,000 mL of hemoperitoneum and ruptured giant thecalutein cysts (Figure 3). The uterus appeared soft and enlarged, although there was no evidence of trophoblastic infiltration (Figure 4). Given the life-threatening hemorrhage, a total abdominal hysterectomy plus bilateral salpingo-oophorectomy was performed, resulting in progressive stabilization. Histopathological analysis confirmed the diagnosis of ruptured bilateral thecalutein cysts and ruled out active gestational trophoblastic neoplasia. During postoperative follow-up, β-hCG levels normalized, and hormone replacement therapy was initiated. At the one-year follow-up, the patient remains asymptomatic and has had no new complications.

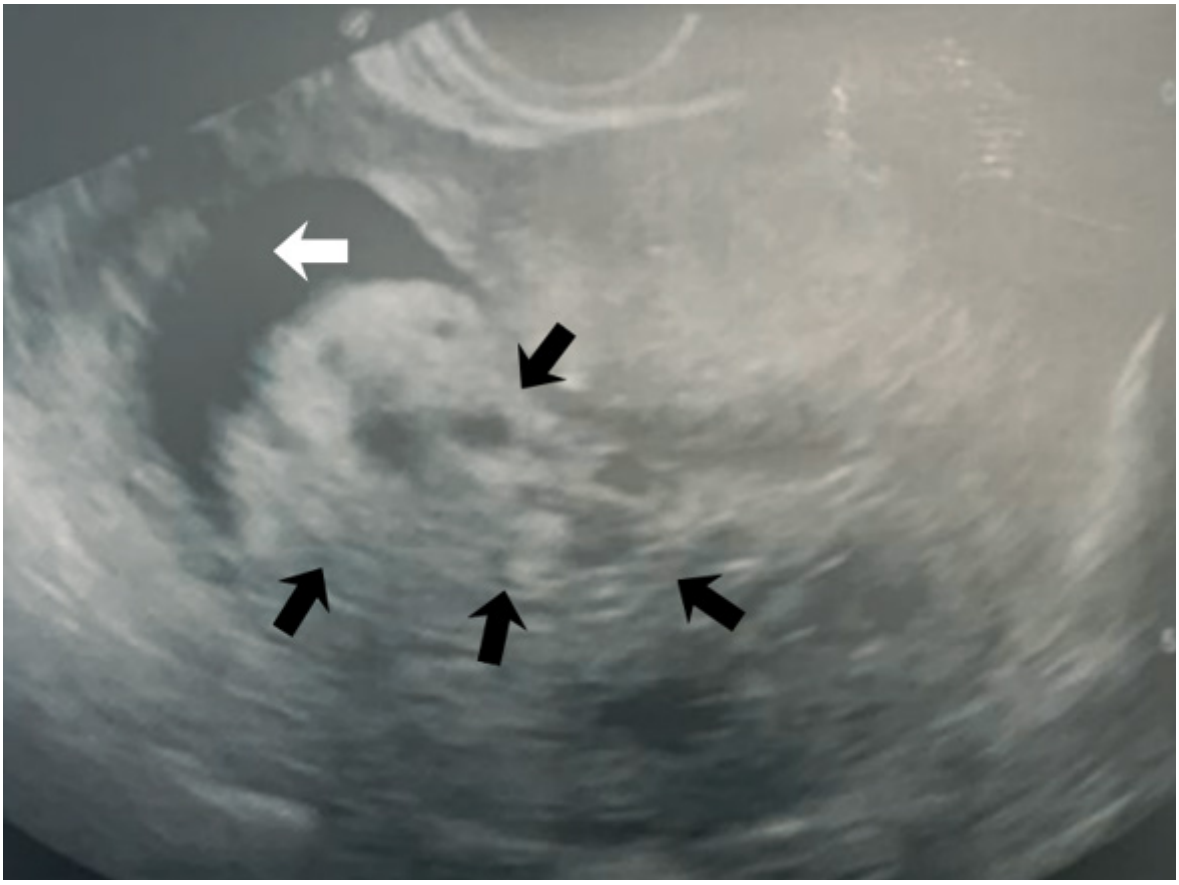

Figure 1. Obstetric ultrasound. Black arrows: Hydatidiform mole. White arrow: Free fluid.

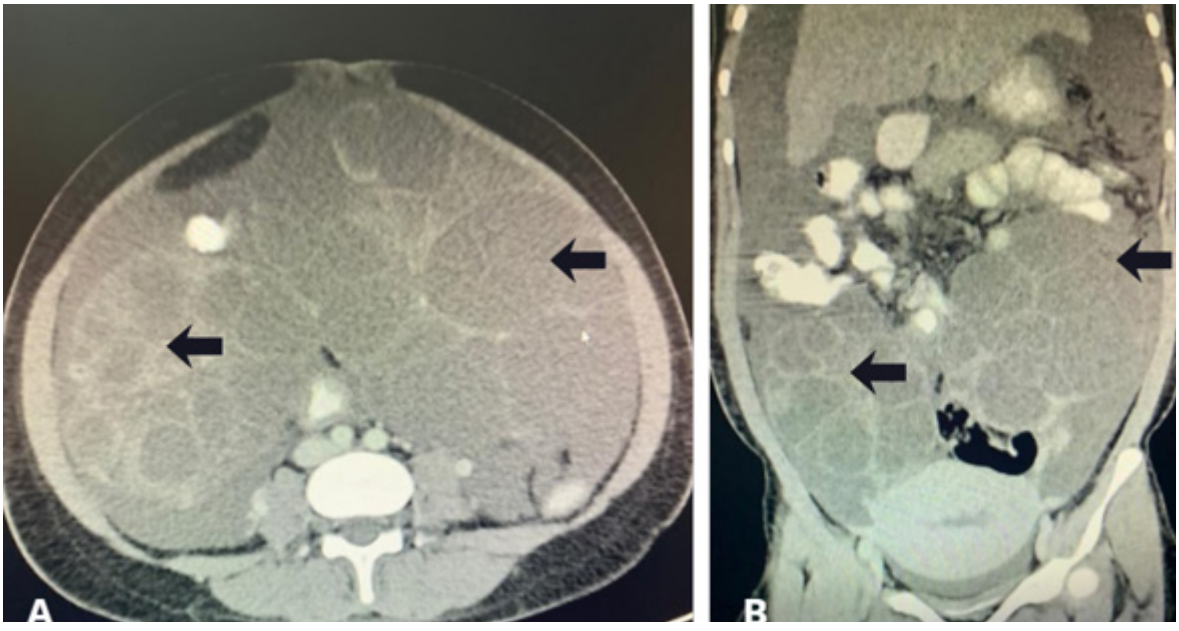

Figure 2. Contrast-enhanced computed tomography; note the giant bilateral adnexal masses (black arrows). A: Axial view; B: Coronal view.

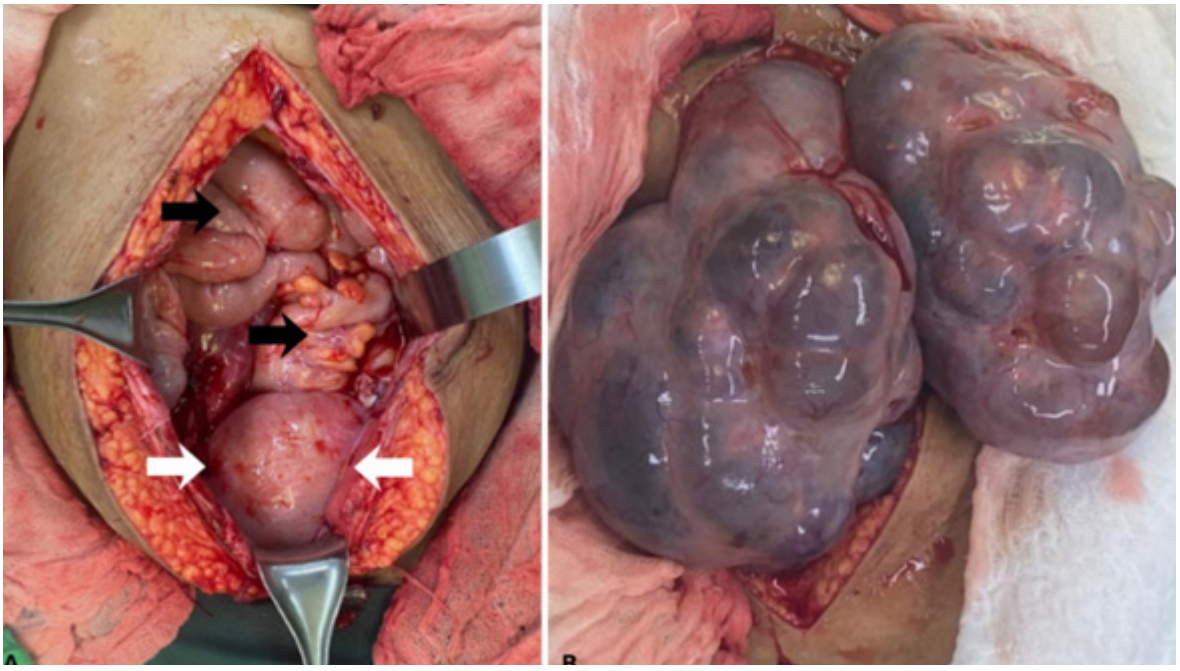

Figure 3. Intraoperative view, A: Abdominal cavity; note significant hemoperitoneum. White arrows: Uterus. Black arrows: Dilated intestinal loops. B: Externalized thecalutein cysts; note some ruptured structures.

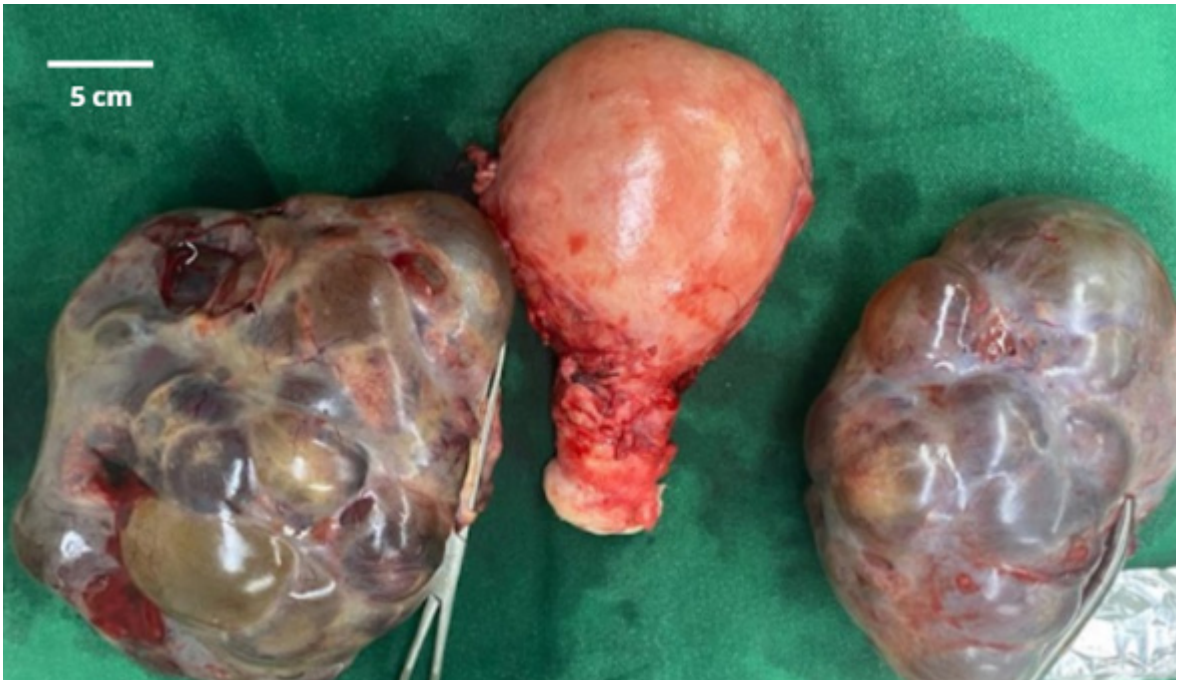

Figure 4. Exposed surgical specimens, uterus, and thecalutein cysts; note the comparative dimensions.

Discussion

Hyperreactio luteinalis (HL) represents an exaggerated physiological response to elevated levels of serum β-hCG or to increased sensitivity of LH/hCG receptors in the inner theca cells[2].

Although its association with GTD is well established, the magnitude and clinical timeline of this specific case place it within an unusual clinical spectrum, where genetic factors appear to play an important role in the pathogenesis[2,8,13,14].

Table 1. Laboratory results upon hospital admission.

Parameter	Result	Unit	Reference	Interpretation
pH	7.21	—	7.35 – 7.45	Metabolic acidosis
pCO_2	25	mmHg	35 – 45	Decreased (respiratory compensation)
pO_2	92	mmHg	80 – 100	Normal
HCO_3-	10	mmol/L	22 – 26	Decreased (metabolic acidosis)
BE	–12	mmol/L	–2 a +2	Severe metabolic acidosis
SaO_2	96	%	95 – 100	Normal
(Hb)	6.8	g/dL	12 – 16	Severe anemia
TSH	0.9	µIU/mL	0.4 – 4.0	Normal
Creatinine	0.9	mg/dL	0.5 – 1.1	Normal
AST (TGO)	26	U/L	10 – 40	Normal
ALT (TGP)	17	U/L	7 – 56	Normal
Total Bilirubin	0.4	mg/dL	0.1 – 1.2	Normal
Serum lactate	3	mmol/L	0.5 – 2.0	Elevated (tissue hypoperfusion)
hCG	24100	UI/L	menor de 5	Elevated

Table 1. The values indicate metabolic acidosis with respiratory compensation, severe anemia, and a significant elevation in beta-hCG. Institutional reference ranges are included. pH: hydrogen ion concentration; pCO_2: partial pressure of carbon dioxide; pO_2: partial pressure of oxygen; HCO^3-: serum bicarbonate; BE: base excess; SaO_2: oxygen saturation; AST: aspartate aminotransferase; ALT: alanine aminotransferase; TSH: thyroid-stimulating hormone; hCG: human chorionic gonadotropin.

A key point to highlight in this report is the development or persistence of HL during the post-molar follow-up period, specifically four weeks after a successful uterine evacuation. Typically, most cases of HL manifest during the third trimester or at term, and the cysts are expected to regress spontaneously as β-hCG levels normalize[2,8,15]. Its onset may go unnoticed and be asymptomatic with spontaneous resolution, as reported by Edell[16]; however, in cases where growth persists, rupture may occur in the post-evacuation period; Rathod et al. described a similar presentation, suggesting that the risk of surgical complications persists as long as residual β-hCG levels stimulate the ovarian stroma[4]. Cyst rupture can be highly symptomatic, leading—as in our case—to hemodynamic instability with a risk of fatality; a similar case was reported by Adjie, which highlights the need for emergency surgical management as a survival strategy[17].

The literature suggests that persistent elevations in estrogen and progesterone during the post-evacuation phase of a molar pregnancy may continue to stimulate ovarian growth[2,8,10]. In this patient, the initiation of combined oral contraceptives following the evacuation may have acted as a contributing factor in an ovarian stroma already sensitized by massive exposure to β-hCG (initial levels of 207,150 IU/L), thereby perpetuating the hyperstimulation despite the decline in hormone levels[18].

The reported cyst sizes (25 cm each) are exceptional; the medical literature has documented few cases of giant thecalutein cysts (defined as those exceeding 15 cm)[17,19]. The 25-cm cysts observed in this case are extraordinarily rare, exceeding the dimensions reported in extensive reviews such as that by Malinowski et al.[2], case series such as that by Watkins[3], and reports of rupture such as that by Kurakula et al.[11]. While smaller cysts are typically asymptomatic and managed with watchful waiting, masses of this size dramatically increase the risk of critical complications such as torsion, rupture, and hemoperitoneum, as demonstrated by Bruno et al., who documented the torsion of a tecalutein cyst[20]. This anatomical disproportion resulted in extreme hemodynamic severity: while other reports of rupture document moderate hemoperitoneum of 600 mL or 300 mL[2,3,11], our patient presented with severe hemorrhage of 3,000 mL, resulting in hypovolemic shock (mean arterial pressure 57 mmHg) and severe metabolic acidosis (base excess BE -12 mmol/L)—a state of decompensation rarely seen in benign ovarian conditions.

Differential diagnosis remains a challenge, as CA-125 levels are often elevated during pregnancy and the postpartum period, reducing its specificity for malignancy; as a result, the condition may mimic neoplasia, leading to delays in diagnosis and management, as reported by Wang and Chen in their studies[21,22].

Delays in appropriate management can lead to preventable oophorectomies, which impact the patient's fertility (23). Consequently, clinical suspicion, the "spoke-like" ultrasound pattern, and the absence of solid components on CT scans remain the most reliable findings for confirming the benign nature of HL[1,8,9].

Finally, although conservative management is the gold standard for HL in order to preserve fertility, this case illustrates a scenario where radical surgery is mandatory; the rupture of giant cysts, which resulted in massive hemoperitoneum of 3,000 mL, triggered severe hypovolemic shock (Hb 6.8 g/dL and metabolic acidosis), warranting a total abdominal hysterectomy plus bilateral salpingo-oophorectomy to achieve hemodynamic control and stabilize the patient[23]. This radical decision deviates from the standard of organ preservation but aligns with damage control protocols for hemorrhagic emergencies with imminent mortality[24,25]. The patient's course following the elimination of the hemorrhagic focus and the source of hormonal stimulation was satisfactory, with a progressive normalization of β-hCG levels, similar to what has been reported in cases of torsion or persistent hyperestrogenism HL. The one-year follow-up showed complete resolution of metabolic and serological parameters, confirming that, despite the initial severity, the prognosis following emergency surgery is excellent once tissue hypoperfusion has been corrected. This outcome underscores the imperative need for rigorous clinical and biochemical monitoring following the evacuation of molar pregnancies to detect atypical, life-threatening developments.

Conclusion

This clinical case is of great medical importance due to the exceptional presentation of a giant *hyperreactio luteinalis*. With dimensions reaching 25 cm per adnexa, it represents an extreme clinical entity, given that the reviewed literature reports very few cases of thecalutein cysts exceeding 15 cm. The severity of this complication lies in its clinical course, in which, despite an apparently successful evacuation of the hydatidiform mole, the patient suffered a spontaneous cystic rupture during post-molar follow-up.

The massive hemoperitoneum of 3,000 mL triggered severe hypovolemic shock, characterized by critical anemia (Hb 6.8 g/dL), metabolic acidosis, and respiratory distress. This outcome underscores that the risk to life persists beyond the initial treatment of gestational trophoblastic disease; therefore, rigorous clinical and serological follow-up is imperative. Furthermore, this case justifies urgent radical surgical intervention in the face of hemodynamic instability as a definitive measure to ensure the patient's survival in the event of complications of this magnitude.

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